The Potential for International Travelers to Transmit Foreign Animal Diseases to US Livestock or Poultry

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Numerous studies have examined the risk of foreign animal diseases (FAD) being introduced to the United States (US). This existing body of literature has focused primarily on risks associated with importation, both legal and illegal, of live animals, germ plasm, and animal products. What has not been looked at extensively is the risk of FAD introduction via the human travelers themselves. Commercial airlines carry 1.4 million persons across international borders every day (CDC 1998). According to the World Tourism Organization, 528 million people vacationed in foreign countries in 1994. Of these, Americans accounted for 47 million travelers. And over 43 million tourists came to the US in 1995 (WTO 1997). Business travelers, including veterinarians, producers, and representatives and regulators of animal and poultry industries, add many millions to that total each year.

This paper explores the issue of disease transmission via human travelers. The purpose is to highlight and summarize what is currently known regarding the potential for human infection and human-to-animal transmission of FAD, focusing on the International Office of Epizootics (OIE) List A diseases.[1] This paper also explores the ability of humans to be biological or mechanical vectors for each disease.

Human to Animal Transmission

When considering the risk that international travelers play in disease transmission, several factors must be considered. First, the traveler must have contact with the disease agent in the country of origin. The traveler must then either become infected with the agent or become mechanically contaminated with it. After arriving in the US, the traveler must come in contact with a susceptible host or vector so the agent can be transmitted to an animal, resulting in disease. For mechanical transmission to occur, the agent must further be able to survive outside the host for a sufficient length of time for the person to travel to the US.

Table 1, "Humans as Potential Biological Carriers of List A Diseases," summarizes existing literature regarding whether humans can be infected with each disease agent, what the incubation period is in humans, and possible modes of transmission from animals to humans. Table 2, "Qualitative Risk of Human to Animal Transmission," focuses on the ability for the disease causing agent to be transmitted from a human to an animal, including relative risks for such transmission to occur. Both biological and mechanical modes of transmission are included for

[1] OIE List A diseases are those transmissible diseases which have the potential for very serious and rapid spread, are of serious socio-economic or public health consequence, and are of major importance in the international trade of animals and animal products. They include avian influenza, Newcastle disease, Rift Valley fever, foot and mouth disease, swine vesicular disease, vesicular stomatitis, classical swine fever, African horse sickness, African swine fever, bluetongue, contagious bovine pleuropneumonia, lumpy skin disease, peste des petits ruminants, rinderpest, and sheep and goat pox.

each List A disease. The risk ratings listed in Table 2 pertain to human-to-animal transmission in general and are not specific to travelers.

Table 3 lists criteria used to develop the risk ratings. It must be emphasized that these risk levels only pertain to risk from human-to-animal transmission. Other modes of transmission such as animal-to-animal or via contaminated animal products would result in significantly different risk ratings for some diseases. It was the purpose of this paper to focus only on human-to-animal transmission. Risk of disease transmission by travelers is addressed in subsequent sections of this paper.

A review of relevant literature centered around four basic questions: 1) Have there been reported cases of humans being infected with the disease causing agent? 2) Can an infected person transmit the agent back to an animal and if so, via what mode of transmission? 3) Can people act as mechanical vectors for the agent? How long can the agent survive outside the host, and under what conditions? 4) For vector borne diseases, is there a competent vector in the US and could that vector become infected from a person? The data gathered from the literature is presented in the appendix, arranged by disease. These summarized highlights of the List A diseases are not intended to be complete reviews of each disease.

Synopsis of Results: Biological Transmission

The first factor to consider when determining the level of risk for biological transmission from humans to animals is whether or not a human can become infected with the disease agent. Cases of human infection have been reported for avian influenza, Newcastle disease, Rift Valley fever, foot and mouth disease, swine vesicular disease, and vesicular stomatitis. The ability of transmission to take place from humans to animals and the mode of that transmission must also be considered. If there are no documented cases of human-to-animal transmission, one can assess human-to-human modes of transmission for possible extrapolation to human-to-animal transmission.

After assessing each List A disease for these factors, the following risk ratings were determined regarding biological transmission of the disease agent from an infected person to an animal.

Risk Rating	Disease
high	none
moderate	none
low	avian influenza, Newcastle disease
negligible	Rift Valley fever, foot and mouth disease, swine vesicular disease, vesicular stomatitis
none	classical swine fever, African horse sickness, African swine fever, bluetongue, contagious bovine pleuropneumonia, lumpy skin disease, peste des petits ruminants, rinderpest, sheep and goat pox

Synopsis of Results: Mechanical Transmission

There are many factors to consider in order to arrive at a risk rating for mechanical transmission. Whether or not a particular agent is infectious is of importance. Agents requiring a vector for transmission are not at risk for mechanical transmission under this study's focus. The amount of the agent shed in secretions and excretions is also important. If a disease-causing agent is shed in doses too low to cause disease in another host, the risk level is much lower then if high amounts of the agent are shed. Many of the viruses causing List A diseases can survive in the environment at room temperature for extended periods of time. This prolonged survivability increases the risk of disease transmission by allowing more time for contact with a susceptible animal to occur. Another factor to consider in mechanical transmission is the type of contact with the infected animal that is required. If an agent can be transmitted via environmental contact, the level of risk is much higher than if direct contact with lesions is required. An example of environmental contact is a person walking onto an infected premises, never touching an animal yet becoming contaminated with the disease agent. The person then walks onto another premises, again never touchs an animal and yet spreads the agent to the new location. For this project, only the person and their clothing were taken into account when assessing the risk of mechanical transmission from a human to an animal. Any animal products or equipment they might be carrying were not considered.

The following risk ratings were determined for mechanical transmission of List A disease agents from a contaminated person to an animal.

Risk Rating	Disease
high	Newcastle disease, swine vesicular disease
moderate	avian influenza, foot and mouth disease, African swine fever
low	vesicular stomatitis
negligible	Rift Valley fever, classical swine fever, lumpy skin disease, peste des petits ruminants, rinderpest, sheep and goat pox
none	African horse sickness, bluetongue, contagious bovine pleuropneumonia

Disease Transmission by International Travelers

The basic factors of disease transmission by international travelers are no different than those for humans in general. These factors include contact with an infected animal, ability to act as a biological or mechanical vector, and contact with and transmission to a susceptible host or vector. However, the case of the international traveler does add some different twists to these factors.

The likelihood of contact with the disease agent varies greatly depending on the traveler's country of origin and the prevalence of disease in that country. It also depends on the traveler's activities. Did the traveler spend their time only in metropolitan areas with little to no opportunity for

contact with livestock? Or did the traveler spend time in rural areas allowing opportunity for contact with livestock? With the increasing amount of ecotourism by US tourists, more people are spending time in rural areas of other countries, thus increasing their opportunity to come into contact with livestock and disease agents of livestock.

Agents which survive outside the host for only short periods of time are unlikely to be transmitted mechanically by the traveler because of the length of time required to travel internationally. Unfortunately, many of the List A diseases can survive outside the host for extended periods of time. This makes it entirely possible for the agent to be carried back to the US on dirty clothing or foot wear and still be viable.

In order for mechanical transmission to occur, a traveler must have contact with feces or other animal excretions containing the disease agent, wear or pack the contaminated items to travel internationally, then wear these same contaminated items to a location where they have contact with animals. While this scenario is entirely possible, for the majority of travelers it is not highly plausible. Travelers originating their travel in another country are unlikely to bring fecal contaminated clothing with them for an international trip. However, for the US originating tourist who spent time in another country and is now returning to the US, and who packed a limited amount of clothing, it is a more probable scenario.

Once in the US, the traveler must come in contact with a susceptible host. It is unknown how many travelers are likely to do so. Those who routinely have direct contact with animals in the US, such as veterinarians, farmers, ranchers, consultants, farm workers, etc., are obviously at greater risk for transmission of diseases then the general traveler.

When considering the risk of humans biologically transmitting diseases, the duration of the travel comes into play. The length of most international air flights is less than the incubation period of many infectious diseases. People incubating diseases can leave the location they became infected, travel to the US, and transmit the disease, before showing any symptoms of infection. A disease can truly move around the globe before the person incubating it has any indication that they are infected.

Conclusion

It is possible for humans to transmit OIE List A diseases to animals in the US. However, for most of the List A diseases, the risk of either biological or mechanical transmission is negligible to none. At high risk for mechanical transmission are Newcastle disease and swine vesicular disease. Avian influenza, foot and mouth disease, and African swine fever are at moderate risk for mechanical transmission while vesicular stomatitis is at low risk for mechanical transmission. Avian influenza and Newcastle disease have a low risk for biological transmission. Mitigating factors brought into account by international travel further reduces the risk of effective human-to-animal transmission of List A diseases.

Table 1: Humans as Potential Biological Carriers of OIE List A Diseases

Disease	Reports of human infection	Incubation period in humans	How humans may become infected
Avian Influenza	Yes	1-3 days	direct contact with infected animals, primarily birds or pigs (at risk: veterinarians, bird owners); potentially from lake or pond water where infected waterfowl are present
Newcastle Disease	Yes	1-2 days	direct contact with infected tissues (at risk: poultry processors, vaccination crews); laboratory exposure; aerosol exposure
Rift Valley Fever	Yes	3-6 days	direct contact with infected animals or tissues (at risk: veterinarians, butchers, meat handlers); via mosquitoes; aerosol in laboratory and slaughterhouse
Foot and Mouth Disease	Yes	2-6 days	drinking raw or pasteurized milk from an infected animal; direct contact
Swine Vesicular Disease	Yes	unknown	direct contact; aerosol; laboratory exposure
Vesicular Stomatitis	Yes	2-6 days	direct contact
Classical Swine Fever African Horse Sickness African Swine Fever Bluetongue Contagious Bovine Pleuropneumonia Lumpy Skin Disease Peste des Petits Ruminants Rinderpest Sheep and Goat Pox	No	not applicable	human infection not reported, with 2 exceptions: (1) mild cases of African horse sickness were recorded in two Middle Eastern men (2) two cases of sheep and goat pox were suspected but not verified

Table 2: Qualitative Risk of Human-to-Animal Transmission

Disease	Possible modes of transmission from humans to animals		Qualitative risk of human-to-animal transmission[2]
	Biological	Mechanical[3]	
Avian Influenza	- respiratory	- droplet or fecal contamination on clothing, shoes, etc - survives 7 days in feces at 20°C, longer in colder temperatures	biological: low mechanical: moderate
Newcastle Disease	- direct contact	- via feces or bird secretions - stable in feces, survives months at 20°C	biological: low mechanical: high
Rift Valley Fever	- via mosquito or other blood feeding insects - may be possible via aerosol	- survives 3 months or more in the environment at room temperature - survives hours in aerosols at 25°C - via mosquito or other blood feeding insects	biological: negligible mechanical: neglibible
Foot and Mouth Disease	- respiratory - direct contact	- virus on shoes, clothing, etc. - stable in the environment for months	biological: negligible mechanical: moderate
Swine Vesicular Disease	- unknown, possibly fecal oral route	- via contaminated clothing, shoes, etc. - survives 4 months or more in feces and infected tissues - resistant to high temperatures	biological: negligible mechanical: high
Vesicular Stomatitis	- via insect vector	- survives several weeks depending on environmental conditions - inactivated at 58°C in 30 minutes	biological: negligible mechanical: low

[2] It must be emphasized that these risk levels only pertain to risk from human-to-animal transmission. There are other modes of transmission such as animal-to-animal or via contaminated animal products that would result in significantly different risk ratings for some diseases.

[3] Mechanical transmission for this study only addressed the possibility of humans acting as a mechanical vector and did not take into consideration any products or equipment they might be carrying. Contamination of the disease agent on clothing, shoes, etc. was considered in assessing risk.

Table 2: Qualitative Risk of Human-to-Animal Transmission

Disease	Possible modes of transmission from humans to animals		Qualitative risk of human-to-animal transmission[2]
	Biological	Mechanical[3]	
Classical Swine Fever	- none	- possible but rare via fecal contamination on clothing, shoes, etc. - survives several days in manure	biological: none mechanical: negligible
African Horse Sickness	- none	- none	none
African Swine Fever	- none	- via clothing, shoes, etc. contaminated with manure - survives 11 days in feces at room temperature - possibly via human carrying infected tick to US	biological: none mechanical: moderate
Bluetongue	- none	- none	none
Contagious Bovine Pleuropneumonia	- none	- none	none
Lumpy Skin Disease	- none	- transmission via fomites has been reported	biological: none mechanical: negligible
Peste des Petits Ruminants	- none	- possibly via manure contaminated clothing, shoes, etc. - survives several hours at 37°C, < 1 hour at 50°C, longer at cooler temperatures	biological: none mechanical: negligible
Rinderpest	- none	- survives a few hours outside the host - can survive as long as 4 days in the presence of moisture	biological: none mechanical: negligible
Sheep and Goat Pox	- none	- survives 6 months in the environment - survives 3 months or more in skin scales	biological: none mechanical: negligible

Table 3: Criteria for Risk Ratings

Mechanical Transmission[4]	
none	- non-contagious virus - no mechanical transmission known
negligible	- virus not highly infectious - mechanical transmission documented - virus survives outside the host for a minimum of a few hours - mechanical transmission minimal or primarily via biting insect - mechanical transmission plays a minor role in disease transmission in the field
low	- virus is infectious - virus survives outside the host for a minimum of 24 hours - virus shed in large enough quantities to transmit disease - mechanical transmission requires direct contact with lesions, or environmental or casual contact - mechanical transmission plays significant role in disease transmission in the field
moderate	- mechanical transmission occurs via environmental or casual contact
high	- virus shed in high quantities - virus survives outside the host for a minimum of one month - mechanical transmission plays a major role in disease transmission in the field
Biological Transmission	
none	- humans can not be infected
negligible	- humans can be infected - human-to-animal transmission biologically possible but not documented - human-to-human transmission biologically possible but not documented
low	- human-to-animal transmission documented - low number of cases reported in humans, or low virus levels in humans, resulting in low transmission opportunity

[4] For a disease to be classified in a particular risk category, it has to meet all requirements of that risk level as indicated in the table, as well has having met or exceeded the requirements of the risk levels up to that point. For example, to be classified as "moderate" risk for mechanical transmission, the disease meets all requirements of the moderate level and has met or exceeded the requirements for the low and negligible categories. A disease classified as "none" does not meet all the requirements of the negligable category, therefore, falls back to the "none" classification.

Appendix

This appendix provides further detail on each of the 15 major diseases reviewed, focusing on

- stability of the disease agent outside the host,
- potential for human infection and disease symptoms in humans, and
- potential for human-to-animal transmission.

Avian Influenza (Fowl Plague)

Avian influenza virus (AIV), like all other influenza viruses, are in the family Orthomyxoviridae. There are three antigenically distinct types of influenza viruses: A, B and C. The causal viruses of avian influenza are type A, which are found in humans, swine, horses, occasionally other mammals, and avian species (Easterday 1984). Type B and C influenza viruses have been found only in humans, except for one report of a type C influenza virus in pigs (Easterday 1984).

Influenza viruses can survive for long periods in cold, moist environments. They are easily inactivated by heat, extremes of pH, and dryness. Virus infectivity is retained in fecal matter for 30-35 days at 4°C and for 7 days at 20°C (Easterday 1984). Influenza viruses have been recovered from lake and pond water where there are large concentrations of waterfowl, though it is uncertain how long the virus can persist in water when infected birds are no longer present. AIVs are rarely sensitive to inactivation by lipid solvents such as detergents.

Influenza as a clinical entity in both humans and animals dates back centuries. In humans the disease affects chiefly the respiratory system and is temporarily incapacitating. Mortality is usually low (Kaplan 1982). Direct transfer of a swine strain and an avian strain from animals to humans, with resultant clinical illness, have been reported but very rarely. In the United Kingdom, AIV was isolated in 1996 from a duck owner who had developed conjunctivitis (Kurtz 1996). A virus similar to AIV was isolated from the blood of a US veterinarian after he had visited the Middle and Far East, though he had no known contact with infected birds (Easterday 1984). Also in the US, an influenza virus with avian characteristics that was isolated from harbor seals was shown to be infectious for humans (Easterday 1984, Kaplan 1982). In 1997, an AIV resulted in disease in 18 humans in Hong Kong, 6 of the cases being fatal. The source of the AIV was infected chickens (ProMED-mail).

The major implication of animal influenza infections for humans is the hypothesis that new strains of AIVs emerge through recombination of influenza viruses in some animal species. Capacity for random reassortment and repackaging of the relevant genomes has been shown to quite readily facilitate production of new viruses. Thus, a reasonable mechanism exists for the origin of “new” human strains in animal species. If genes from human strains are involved in the recombination, a basis for interspecies transfer and subsequent perpetuation in humans is provided.

A wealth of circumstantial evidence exists to suggest interspecies transfer of AIVs in a swine-avian-human chain. Like birds, swine may provide a reservoir for influenza strains that, either by themselves or following recombination with human strains, produce influenza pandemics in humans. The strains responsible for the 1957 and 1968 human pandemics were reassortants incorporating both human and avian influenza viruses, which may have arisen in pigs (Zhou 1996). In the US, a 1976 outbreak among soldiers at Fort Dix, New Jersey was attributed to a swine influenza virus (Webster 1995). In Europe, transmission of AIV to swine resulted in the appearance of human-avian reassortment influenza viruses in pigs in Italy and in children in the Netherlands (Webster 1995).

While human-to-animal transmission of AIV has been documented, the data is somewhat vague about the direction and spread of the virus. In one Japanese outbreak, pigs clearly contracted Hong Kong flu from humans (Sabine 1993). Human influenza virus can multiply in ducks but does not seem to be transmitted among them. There is firm evidence that pigs can become infected by human and avian influenza virus. Pigs may transmit both types of virus to other pigs as well as back to the original hosts (Sabine 1993, Kaplan 1982).

Humans may also potentially transmit AIV to other animal species. Strains of virus believed to be derived from human sources have caused disease in chickens and calves (Ritchie 1988). Antibodies related to the H component of an equine 2 subtype were detected in people alive around 1890, indicating the strain involved in human infections at that time may have been related to strains infecting horses (Kaplan 1982, Ritchie 1988).

Newcastle Disease

Newcastle disease is caused by a paramyxovirus that is stable outside the host. Although warm temperatures and sunlight speed inactivation of Newcastle disease virus (NDV), the virus is quite resistant to changes in pH and heat. As temperatures get lower from 37°C, the virus survives longer in the environment. Years are required to inactivate the virus at 8°C and freezing does not inactivate the virus (Beard 1984). As temperatures rise above 37°C, the virus survives for longer periods of time. It has remained stable at 50°C for 134 days (Ritchie 1995). NDV can remain active in moist soil for 22 days, on feathers at 20°C for 123 days and in lake water for 19 days (Ritchie 1995). The virus is excreted in high concentrations in the feces, which provides a stable medium for virus survival outside the host. It has been isolated from buried carcasses after 121 days (Ritchie 1995).

Newcastle disease in humans has an incubation period of 1 to 2 days. It primarily causes a conjunctivitis lasting 3 to 4 days with the virus being present in ocular fluids from 4 to 7 days. Recovery is uneventful with no treatment required. Generalized infection has been reported causing flu-like symptoms lasting 3 to 4 days (Hanson 1975, Ritchie 1995). The virus has been isolated from nasopharyngeal washings, saliva, blood, and urine. Subclinical disease in humans is also possible.

Typically, transmission to humans is via direct contact with infected chicken tissues in poultry processing plant workers or vaccination crews. Laboratory workers have also been infected

(Hanson 1975). There have been no reported clinical cases of Newcastle disease in workers caring for live chickens and no known transmission via handling or consuming poultry products. Although NDV has been isolated from numerous pet bird species, there have been no reports of transmission to people. This may, however, be due to the clinical similarities to influenza and the fact that recoveries are typically uneventful, resulting in a lack of disease diagnosis and reporting. Transmission to humans via an aerosol route (birds beating their wings in a dusty environment resulting in airborne viral particles) can result in a generalized viral infection (Ritchie 1995).

Humans infected with NDV can transmit the virus to live birds. People of concern are animal or disease specialists who provide consulting services, traveling from country to country. However, multiplication of NDV in humans is considered to be limited, resulting in infrequent transmission from an infected person under natural conditions. Mechanical transmission of NDV can occur by insects, rodents, and humans. Movement of people and equipment is considered to be a significant factor in the mechanical spread of disease during an outbreak situation (Alexander 1988).

Rift Valley Fever

The causal agent of Rift Valley fever is the Rift Valley fever virus (RVFV) in the family Bunyaviridae and the genus *Phlebovirus*. The virus infects and causes disease in a wide range of host species including cattle, sheep, goats, and humans. Some animals such as swine, chicken, and rabbits are refractory to the disease.

The virus is relatively stable outside the host. In one report, RVFV remained viable in the environment for more than three months, after which time, a laboratory technician became infected with the virus. The technician contracted the disease after scraping and painting the walls and floor of an animal room which had been used in a study of RVFV three months prior (Van Velden 1977). Frozen virus can remain infective for 8 years (Easterday 1965). In aerosols, the virus has a half-life greater than 77 minutes at 25°C and 30 percent relative humidity (Hoogstraal 1979).

While humans can become infected with RVFV, the mode of animal-to-human transmission is not definitively known. Most literature reviewed for this report indicated that the primary mode of transmission to humans is via contact with infected animals or animal tissues, such as during necropsy or butchering. Veterinarians, butchers, and meat handlers are reported to have become infected by handling carcasses or meat from infected animals (Beneson 1990, Wilson 1994). One source considered mosquito transmission to be the primary zoonotic mode of transmission. (Wilson 1994). *Aedes* mosquitoes are competent vectors for RVFV and have the potential of transmitting the virus in the Americas. Some *Culex* species are also competent vectors for RVFV and they do feed on humans as well as animals (Wilson 1994). Aerosol transmission of RVFV has also occurred in laboratory and slaughterhouse workers (Sabine 1993, Benenson 1990). Primary risk factors for human infection have been reported to be caring for humans infected with RVFV, assisting animals during births/abortions, and treating sick animals (Wilson 1994).

The incubation period for RVFV in humans is three to six days. Disease can manifest in four different forms:

1) influenza-like symptoms including fever, malaise, anorexia, and prostration with an uneventful recovery in 4 to 7 days;
2) meningoencephalitis;
3) retinitis with potential for permanent loss of central visual acuity; or
4) hemorrhagic disease characterized by profuse bleeding, jaundice, hematemesis, melena, and/or petechiae, possibly leading to death.

Human-to-human transmission of RVFV appears possible but there have been no documented occurances of such transmission reported in the literature. One paper reported the isolation of virus from nasopharyngeal secretions, indicating the potential for human-to-human transmission via droplets (Abdel-Wahab 1978). Another study found no clustering of human infections within living compounds during an outbreak, indicating that the casual contact of daily living activities is not sufficient to transmit the virus. However, caring for individuals infected with RVFV did result in an increased risk of infection (Wilson 1994).

Human-to-animal transmission appears to require biological or mechanical transmission by an insect vector. Humans infected with RVFV have enough viremia to infect multiple mosquito species. Experimentally, numerous North American mosquito species are competent biologic vectors of RVFV (Gargan 1988, Turell 1988). Sand flies have also transmitted the virus in laboratory studies, and various blood feeding insects can transmit RVFV mechanically (Hoch 1985). Given that competent insect vectors appear critical for any human-to-animal transmission of RVFV, introduction of RVFV into the US would pose the greatest risk during spring and early summer and in geographic regions where vectors are abundant.

Foot and Mouth Disease

Foot and mouth disease virus (FMDV), a picornavirus of the genus *Apthovirus*, has been widely studied for its strong environmental stability. There are multiple sub-types of FMDV (at least 65 divided into 7 antigenic types). Generally, infection with one type fails to provide immunity to infection with other types.

FMDV is very hearty, surviving freezing temperatures. It has been found to be viable in contaminated milk after pasteurization at 72°C for 15 seconds (Pirtle 1991). The virus can survive for extended periods outside the host in protected locations. FMDV has been recovered from cattle stalls 14 days after removal of infected cattle, from urine after 39 days, from soil after 28 days in autumn and after 3 days in summer, and from dry hay at 22°C after 20 weeks storage (Pirtle 1991). The virus is inactivated by sunlight, extremes in pH, and high temperatures.

Human infections do occur, but are extremely rare. The incubation period is from two to six days and the course of disease is short and uncomplicated. Symptoms include fever, vomiting, a sense of heat and dryness in the mouth, and small vesicles on the lips, tongue, and oral mucosa. Vesicles can also occur on the hands or feet. Asymptomatic infections are possible.

Probably the most important route of transmission to humans is ingestion of FMDV infected milk, especially raw milk. Close contact with infected animals can also result in disease transmission, especially in outbreak situations when livestock mortality is high and large amounts of virus are shed into the environment. Groups at risk include herdsmen, veterinarians, technicians, and consumers of raw dairy products.

While clear records of infection with FMDV in humans do exist with confirmed cases in several nations of Eastern and Western Europe, Africa, and South America, the number of confirmed cases on record is less than fifty (Gustafson 1975, Betts 1952, Timoney 1988, USDA:APHIS 1994, Dlugosz 1943). No cases of human infection were recognized during the most recent FMD outbreaks in Mexico and the US, although countless people had close contact with the virus. It has been noted that "the number of credible cases in relation to the number of persons exposed is infinitesimal" (Betts 1952).

Humans can play a role in the transmission of FMDV. The virus can be carried by clinically affected humans for up to approximately 14 days after the onset of the disease. Humans can inhale the virus, trapping it in the respiratory tract for as long as 36 hours (Hyslop 1973). It can then be expelled in the saliva or breath, thus potentially serving as a source of infection to susceptible animals. It is also possible for humans to asymptomatically spread FMDV; however, failed experiments indicate that it is highly unlikely that this mode of transmission would be a source of infection for animals (Callis 1982).

The lack of human cases over the years adds to the general conclusion from human susceptibility studies that humans are not very susceptible to the virus and, at most, play only a minor role in the biological transmission of FMDV (Gustafson 1975). In addition, there is no evidence that spread from human-to-human has occurred, although theoretically it seems possible.

The most important form of transmission of FMDV from humans to animals is mechanical, as the virus can persist on clothing, shoes, or luggage for at least 9 weeks (Cottral 1969). Human travelers are definitely a possible source of infection via this mode of transmission.

Swine Vesicular Disease

Swine vesicular disease virus (SVDV) is classified in the family Picornaviridae, genus *Enterovirus*. Swine and humans are the only known hosts susceptable to natural infection by SVDV. SVDV is serologically related to coxsackie B5 virus, a common enterovirus of humans which can also produce an inapparent infection in pigs.

SVDV remains stable under a variety of environmental conditions. Temperatures greater than 60°C are necessary to inactivate the virus by heat alone. It is stable in feces and infected tissue kept at ambient or higher temperatures for as long as four months or more (Graves 1975). The stability of SVDV outside the host can be surprising. For example, it has been isolated from the surface and gut of earthworms collected from the soil above the buried carcasses of infected pigs (Callis 1982).

Potential for human infection is very real for those working with SVDV or SVDV-infected pigs. Animal-to-human transmission can occur by direct contact with skin lesions or feces of infected pigs or via airborne excretion of the virus from the skin lesions of an infected animal. High levels of antibody to SVDV have been found in laboratory workers (Graves 1975).

Human infection with SVDV does not typically result in vesicle formation. Symptoms are similar to those caused by the Coxsackie group of human enteroviruses, including fever, sore throat, headache, and vomiting.

Vesicular Stomatitis

Vesicular stomatitis is caused by an arbovirus. The vesicular stomatitis virus (VSV) is relatively stable outside the host, surviving several weeks depending on environmental conditions and the medium in which the virus is present. VSV is moderately resistant to heat and when frozen, has remained viable for several years (Watson 1981).

Humans can become infected by sustained, intensive exposure to VSV infected livestock. Disease in humans is influenza-like, characterized by fever, headache, malaise, nausea, pharyngitis, cervical adenopathy, and vesicular lesions on the oral mucosa. Viremia lasts approximately 24 hours before and after the onset of fever.

During the 1982-83 vesicular stomatitis outbreak in Colorado, seroprevalence rates of exposed persons were determined and risks and probability of various modes of animal-to-human transmission were quantified (Reif 1987). The seroprevalence rate for VSV was found to be 13 percent among veterinarians (private, university, and government) and veterinary students who were exposed to VSV. Relative risks for VSV transmission from an infected animal to veterinary personnel were estimated to be highest when an infected animal sneezed in the veterinarian's face, when the veterinarian had open wounds on a hand or arm, or when saliva from an infected animal touched the veterinarian's eye. Examining oral lesions by opening infected animals' mouths and examining horses versus cattle were estimated to be of lower risk. It was concluded that close, direct contact with an infected animal was needed for animal-to-human transmission of VSV.

Biological transmission of VSV from humans to animals appears possible primarily through a competent insect vector rather than by direct contact due to the rarity of vesicle formation in humans. Insects that bite infected humans become infective approximately 7 days later. They remain infective for the rest of their normal life span of approximately one month. There is no evidence of human-to-human transmission. Several studies have found no evidence of infection in family members of clinically affected VSV infected animal workers (Patterson 1958, Ellis 1964).

Classical Swine Fever

The causative viral agent of classical swine fever is in the pestivirus group of the family Flaviviridae. Swine are the only natural host. Outside of the host, the classical swine fever virus (CSFV) tends to be resistant in the environment, especially if kept cold or frozen. The virus can

survive for periods of time in pens and manure, but is inactivated by sunlight. In pork and pork products, it remains infective for months or years if the product is frozen (Terpstra 1987).

The primary mode of animal-to-animal transmission of CSFV is via direct contact. Transmission to pigs has been accomplished experimentally by oral, nasal, aerosol, conjunctival, genital, and various parenteral routes. It is thought that all of these routes also occur naturally (Terpstra 1987). High amounts of virus are present in blood, tissues, and oral fluids (Terpstra 1987). The amount of virus shed in feces and urine is low (Terpstra 1987). Humans are not susceptible to the classical swine fever virus.

Mechanical transmission by contaminated instruments can readily spread the virus and it has been experimentally transmitted by horseflies (Tidwell 1972). While mechanical transmission by other means such as contaminated clothing, footwear, pets, and rodents is possible, it is of little significance due to the amount of virus present usually being less than the infective dose (Terpstra 1987).

African Horse Sickness

The causative agent of African horse sickness is a pantropic arbovirus of the family Reoviridae, genus *Orbivirus*. African horse sickness virus (AHSV) can withstand prolonged periods of time outside the horse at low temperatures. At very warm temperatures the virus is inactivated. For example, at 37°C it remains infective for 2 weeks (USDA:APHIS 1992) while at 45°C the virus only remains infective for 6 days (Howell 1968).

There is no evidence that humans can become infected with field strains of AHSV, either through contact with infected animals or from working in laboratories. The only recorded human case of the disease is from the Middle East where two men reportedly experienced mild fever and later developed an antibody titer to the virus (Timoney 1988, USDA:APHIS 1992). It has been shown that certain neurotropic vaccine strains can cause encephalitis and retinitis in humans following transnasal infection (Swanepoel 1992). Serological evidence has been found of accidental aerosol infection of 4 humans who were working with vaccine strains (serotypes 1 and 6) (Coetzer 1994). These persons suffered from non-fatal encephalitis and chorioretinitis which resulted in permanent partial loss of vision and blindness. There is no known role that humans play in transmission of the disease.

AHS is considered to be a non-contagious disease. No transmission by contact, inhalation, or ingestion is known to occur. AHSV has been shown to be transmitted biologically by midges (*Culicoides* spp.), the most significant appearing to be *Culicoides imicola.* However, other species such as *C. variipennis* are common in many parts of the US and should also be considered as a potential vectors (USAHA 1992). Mechanical transmission by several species of ticks is also possible (Coetzer 1994, Timoney 1988). The disease has a seasonal occurrence, primarily in the late summer, disappearing quickly after frosts come. Its prevalence is therefore influenced by climatic and other conditions which favor the breeding of *Culicoides* spp. One source noted that "with modern transport no country is safe from infection as the vector can survive plane trips" (Callis 1982).

The dog has long been known to be susceptible to experimental infection with AHSV as well as easily becoming infected by eating infected horse meat. It is, however, very unlikely that dogs become infected by insect bites and it is generally accepted that dogs play no role in the spread or maintenance of the disease (Coetzer 1994, Timoney 1988).

African Swine Fever

Swine are the only natural host for the African swine fever virus (ASFV). It does not infect humans. The viral agent is highly resistant to environmental conditions. It can be isolated from serum or blood that has been kept at room temperature for 18 months or from refrigerated serum or blood after 6 years (Plowright 1994). The virus is present in feces for 11 days at room temperature (Plowright 1994).

Soft ticks (*Ornithodoros* spp.) have been identified as the reservoir for ASFV. Infected ticks can survive for months or years in the absence of pigs by feeding on other animals or humans. In one report, infected ticks were still present in the local environment eight months after the end of an outbreak (Haresnape 1989). Infected ticks can transmit ASFV to reintroduced, disease-free swine after the disease has been cleared from an area. One infected tick has enough virus to infect and cause disease in a pig (Plowright 1970). *Ornithodoros coriaceus*, indigenous to the US, has been experimentally infected with ASFV and was capable of transmitting the virus. However, this species is unlikely to be a permanent reservoir as the virus was not maintained through the egg to the next generation of larvae (Groocock 1980). *O. turicata*, collected in Florida, is also experimentally able to transmit the ASFV (Hess 1987).

ASFV can also be mechanically transmitted on vehicles contaminated with feces from infected animals. Experimentally, mechanical transmission by flies has occurred (Mellor 1987).

Bluetongue

Bluetongue virus (BTV) is classified in the family Reoviridae, genus *Orbivirus*. The virus does not exist outside the live host animal or insect vector, nor is it viable in meat or animal products. In blood and tissue specimens, the BTV is very stable at a wide range of temperatures, becoming inactivated at high temperatures. When freeze-dried in certain mediums, BTV can survive almost indefinitely at room temperature (Verwoerd 1994). It is readily inactivated by a number of common disinfectants.

The primary mode of BTV transmission is by *Culicoides* spp., biting midges or gnats. The principal vector in the US is *Culicoides variipennis*. The vector reaches maximum transmission potential 10-14 days after taking a blood meal from a viremic animal (Callis 1982). Vector competence studies have shown that the bite of a single infected midge is sufficient to transmit BTV to a susceptible animal (Verwoerd 1994). Ticks may also be capable of mechanically or biologically transmitting BTV, however their role appears minimal (USAHA 1992).

BTV can be transmitted by transfer of blood from an infected animal, from a viremic dam to a fetus, or by poor management practices such as using the same needle or contaminated equipment on a number of animals. There is no evidence that BTV causes disease in humans or can be naturally transmitted to humans. Humans also do not appear to play any significant role as either biological or mechanical vectors, with the exception of poor management practices.

Contagious Bovine Pleuropneumonia

The causitive agent for contagious bovine pleuropneumonia is *Mycoplasma mycoides* subspecies *mycoides*. Cattle are the only hosts for the organism. Animal-to-animal transmission is primarily respiratory by the inhalation of droplets produced by sneezing and coughing. Close contact between animals is required as the organism is not highly infectious. *Mycoplasma mycoides* has been cultured from the urine of infected cattle, therefore, it is thought that the organism may be transmitted via the urine (Masiga 1972).

Mycoplasma mycoides does not survive outside the host for more than a few days (USDA:APHIS 1984). Mechanical transmission does not appear to play a role in disease transmission, nor do humans.

Lumpy Skin Disease

Lumpy skin disease in cattle is caused by a virus belonging to the family Poxviridae, genus *Capripoxvirus*. The lumpy skin disease virus (LSDV) is remarkably stable and resistant to physical and chemical agents. LSDV has been isolated from dry necrotic skin lesions in a salted fresh hide and from an air dried hide kept at room temperature for 18 days (USDA:APHIS 1984). The virus has also been shown to persist in necrotic skin nodules for up to 33 days (Barnard 1994, Weiss 1968).

The mode of transmission of LSDV has not been established, although circumstantial evidence suggests that biting insects, probably mosquitoes, play a major role in disseminating infection (USDA:APHIS 1984, Barnard 1994). The spread of LSDV usually follows road, rail, or cattle migration routes, and infection is more prevalent in wet summer and autumn months, particularly in low-lying areas where mosquitoes breed (Barnard 1994).

It is believed that saliva of infected animals might spread the disease where common feeding and drinking troughs are used (Callis 1982). However, if direct contact plays a role in the spread of LSDV, it is a minor one. Spread by direct contact and fomites has been reported in Africa (USDA:APHIS 1984, Barnard 1994), but appears to play a minor role in the epidemiology of the disease. There are no reports of LSDV infecting humans, nor is there evidence that the virus causes disease in humans.

Peste des Petits Ruminants

Peste des petits ruminants virus (PPRV) is a morbillivirus. Few studies have looked at the survival of infectious PPRV outside the host. One laboratory study showed the virus survived only a few hours at 37°C and less than one hour at 50°C (Rossiter and Taylor 1994). The virus

survived longer in colder temperatures, with extremes in heat or pH rapidly inactivating the virus. Humans are not known to be susceptible to the virus.

The primary mode of transmission is through inhalation of aerosols produced by sneezing and coughing and direct contact with viral laden ocular, nasal, and oral secretions and feces. Of lesser importance is indirect transmission via fomites contaminated with secretions and excretions of an infected animal. Mechanical transmission is possible, however, given the short time periods the virus can survive outside the host, this would be a transmission route of minor importance.

Rinderpest

Rinderpest is also caused by a morbillivirus. The virus typically survives only a few hours outside the host, but may persist up to 4 days especially in the presence of moisture (Rossiter 1994). It is rapidly inactivated by light and UV radiation. The virus is can be found in basically all tissues and fluids from an infected animal (USAHA 1992). Transmission is primarily via droplets, requiring close contact. It can also be transmitted by contaminated feed. There is no evidence that rinderpest virus can cause disease in humans.

Sheep and Goat Pox

The causative agent for sheep and goat pox is *Capripoxvirus*. This virus can survive for prolonged periods in the environment, however, it is inactivated by direct sunlight. The virus is viable in wool for two months and on contaminated premises for six months (Callis 1982). It can survive for at least three months in scab material, which can fall onto pasture plants or the soil, contaminating the environment of an infected animal (Kitching 1985).

The mode of transmission is unknown but is hypothesized to be spread by inhalation of virus contaminated aerosols or dust. It is also hypothesized to be spread by inhalation of airborne droplets or mechanically by biting insects (Munz 1994, Kitching 1985). Fairly intimate contact is thought to be required for transmission, as the virus is not highly infectious. The virus has been isolated from nasal and conjunctival swabs (Kitching 1985); however, if aerosol transmission does take place, very large doses of the virus appear to be required. *Capripoxvirus* has been transmitted experimentally between sheep using the fly *Stomoxys calcitrans* as a vector (Kitching 1986). The virus has also been transmitted experimentally in the absence of biting insects, indicating that insects are not required for transmission (Kitching 1985).

There is no conclusive evidence that sheep and goat pox virus can infect humans. There have been two isolated reports of possible human infection; however, virus was not isolated in either case (USAHA 1992). In one case, diagnosis was based solely on clinical signs.

REFERENCES

Abdel-Wahab, KSE; El Baz, LM (1978) Rift Valley Fever Infections in Egypt. *Transactions of the Royal Society of Tropical Medicine and Hygeine,* **72(4)**, 392-396.

Alexander, DJ (1988) *Newcastle Disease*. Kluwer Academic Publishers, Boston, 256-272.

Barnard, BJH; Munz, E; Dumbell, K; et al (1994) Lumpy skin disease. *Infectious Diseases of Livestock, Vol. 1*, Coetzer, JAW; Thompson, GR; Tustin, RC eds. Oxford University Press, New York, 604-612.

Beard, CW; Hanson, RP (1984) Newcastle Disease. *Diseases of Poultry,* Hofstad, MS ed. Iowa State University Press, Ames.

Beneson, AS (ed) (1990) *Control of Communicabe Diseases in Man* American Public Health Association, Washington, DC.

Betts, A (1952) The Sucesptibility of Man to the Virus of Foot and Mouth Disease. *The Veterinary Record,* **64(43)**, 640-641.

Callis, JJ; Dardiri, AH; Ferris, DH; et al (1982) *Illustration Manual for the Recognition and Diagnosis of Certain Animal Diseases.* Mexico-United States Commission for the Prevention of Foot and Mouth Disease.

CDC (1998) Proceedings of the International conference on emerging infectious diseases, 21.

Coetzer, JAQ; Erasmus, BJ (1994) African horsesickness. *Infectious Diseases of Livestock, Vol. 1*, Coetzer, JAW; Thompson, GR; Tustin, RC eds. Oxford University Press, New York, 460-475.

Cottral, GE (1969) Persistence of foot-and-mouth disease virus in animals, their products and the environment. *Bulletin of the International Office of Epizootics,* **71**, 549-568.

Dlugosz, H (1943) Foot and Mouth Disease in Man. *British Medical Journal*, **1**, 189-190.

Easterday, BC (1965) Rift Valley fever. *Adv. Vet. Sci*, **10**, 65-127.

Easterday, BC; Beard, CW (1984) Avian Influenza. *Diseases of Poultry,* Hofstad, MS ed. Iowa State University Press, Ames.

Ellis, EM; Dendall, HE (1964) The Public Health and Economic Effects of Vesicular Stomatitis in a Herd of Dairy Cattle. *Journal of the American Veterinary Medical Association,* **144(4)**, 377-380.

Gargan II, TP; Clark, GG; Dohm, DJ; et al (1988) Vector potential of selected North American mosquito species for Rift Valley fever virus. *American Journal of Tropical Medical Hygiene,* **38(2)**, 440-446.

Graves, JH; McKercher, PD (1975) Swine Vesicular Disease. *Diseases of Swine*, Dunne, HW; Leman, AD eds. Iowa State University Press, Ames.

Groocock, CM; Hess, WR; Gladney, WJ (1980) Experimental Transmission of African Swine Fever Virus by *Ornithodoros coriaceus,* and Argasid Tick Indigenous to the United States. *American Journal of Veterinary Research*, **41(4)**, 591-594.

Gustafson, DP (1975) Foot and Mouth Disease. *Diseases Transmitted from Animals to Man*, Hubbert, WT; McCulloch, WF; Schnurrenberger, PR eds. Charles C. Thomas Publisher, Illinios.

Hanson, RP (1975) Paramyxovirus infections. *Diseases Transmitted from Animals to Man* (6th Ed), Hubbert, WT; McCulloch, WF; Schnurrenberger, PR eds. Thomas Books, Illinois, 851-858.

Haresnape, JM; Wilkinson, PF (1989) A study of African swine fever virus infected ticks (*Ornithodoros moubata*) collected from three villages in the ASF enzootic area of Malawi following an outbread of the disease in domestic pigs. *Epidemiol Infect*, **102(3)**, 507-522.

Hess, WR; Endris, RG, Haslett, TM; et al (1987) Potential Arthropod Vectors of African Swine Fever Virus in North America and the Caribbean Basin. *Veterinary Parasitology*, **26**, 145-155.

Hoch, AL; Gargan II, TP; Bailey, CL (1985) Mechanical transmission of Rift Valley fever virus by *Hematophagous diptera*. *American Journal of Tropical Medical Hygiene*, **34(1)**, 188-193.

Hoogstraal, H; Meegan, JM; Khalil, GM; et al (1979) The Rift Valley fever epizootic in Egypt 1977-78. Ecological and entomological studies. *Trans R Soc Trop med Hyg*, **73**, 624-629.

Howell, PG (1968) African Horsesickness. *Handbuch der Virusinfektionen bei Tieren*, Röhrer, HH ed. Fischer, Jena, 593-625.

Hyslop, NStG (1973) Transmission of the virus of foot and mouth disease between animals and man. *Bulletin of the World Health Organization*, **49**, 577-585.

Kaplan, MM. (1982) The Epidemiology of Influenza as a Zoonosis. *The Veterinary Record*, **110,** 395-399.

Kitching, RP; Mellor, PS (1986) Insect transmission of capripoxvirus. *Research in Veterinary Science*, **40(2)**, 255-258.

Kitching, RP; Taylor, WP (1985) Transmission of capripoxvirus. *Research in Veterinary Science*, **39(2)**, 196-199.

Kurtz, J; Manvell, RJ; Banks, J (1996) Avian Influenza Virus Isolated from a Woman with Conjunctivitis. *The Lancet*, **348**, 901-902.

Masiga, WN; Windsor, RS; Read, WCS (1972) A New Mode of Spread of Contagious Bovine Pleuropneumonia. *The Veterinary Record*, **90(9)**, 247-248.

Mellor, PS; Kitching, RP; Wilkinson, PJ (1987) Mechanical transmission of capripox virus and African swine fever virus by *Stomoxys calcitrans*. *Res Vet Sci*, **43(1)**, 109-112.

Munz, E; Dumbell, K (1994) Sheeppox and goatpox. *Infectious Diseases of Livestock, Vol. 1*, Coetzer, JAW; Thompson, GR; Tustin, RC eds. Oxford University Press, New York, 613-615.

Patterson, WC; Mott, LO; Jenney, EW (1958) A Studay of Vesicuar Somatitis in Man. *Journal of the American Veterinary Medical Association*, **133(1)**, 57-62.

Pirtle, EC; Beran, GW (1991) Virus Survival in the Environment. *Revue Scientifique et Technique (International Office of Epizoonotics)*, **10(3)**, 733-748.

Plowright, W; Perry, CT; Peirce, MA; et al (1970) Experimental infection of the Argasid tick, *Ornithodoros moubata porcinus*, with African swine fever virus. *Arch. ges. Virusforsch*, 31, 33-50.

Plowright, W; Thomson, GR; Neser, JA (1994) African swine fever. *Infectious Diseases of Livestock, Vol. 1*, Coetzer, JAW; Thompson, GR; Tustin, RC eds. Oxford University Press, New York, 567-599.

ProMED-mail, http://www.healthnet.org/programs/promed.html#archives.

Reif, JS; Webb, PA; Monath, TP; et al (1987) Epizootic Vesicular Stomatitis in Colorado, 1982: Infection in Occupational Risk Groups. *American Journal of Tropical Medicine and Hygeine*, **36(1)**, 177-182.

Ritchie, BW; Carter, K (1995) *Avian Viruses, function and control*. Wingers Publishing, Inc., Florida.

Ritchie, BW; Dreesen, DW (1988) Avian Zoonoses: Proven and Potential Diseases. Part II. Viral, Fungal, and Miscellaneous Diseases. *Compendium Small Animal*, **10(6)**, 688-689.

Rossiter, PB (1994) Rinderpest. *Infectious Diseases of Livestock, Vol. 2*, Coetzer, JAW; Thompson, GR; Tustin, RC eds. Oxford University Press, New York, 735-757.

Rossiter, PB; Taylor, WP (1994) Peste des Petits Ruminants. *Infectious Diseases of Livestock, Vol. 2*, Coetzer, JAW; Thompson, GR; Tustin, RC eds. Oxford University Press, New York, 758-765.

Sabine, M. (1993) Tropical Health: Viral Zoonoses. *The Medical Journal of Australia*, **158**, 754-760.

Swanepoel, R; Erasmus, BJ; Williams, R; et al (1992) Encephalitis and chorioretinitis associated with neurotropic African horsesickness virus infection in laboratory workers. Part III. Virological and serological investigations. *South African Medical Journal*, **81**, 458-461.

Terpstra, C (1987) Epizootiology of swine fever. *The Veterinary Quarterly*, **9 (suppl 1)**, 50S-60S.

Tidwell, MA; Dean, WD; Tidwell, MA; et al (1972) Transmission of hog cholera virus by horseflies (Tabanidae: Diptera). *American Journal of Veterinary Research*, **33(3)**, 615-622.

Timoney, JF; Gillespie, JH; Scott, FW; et al (1988) *Hagan and Bruner's Microbiology and Infectious Diseases of Domestic Animals*. Cornell University Press, New York.

Turell, MJ; Bailey, CL; Beaman, JR (1988) Vector Competence of a Houston, Texas Strain of *Aedes albopictus* for Rift Valley Fever Virus. *Journal of the American Mosquito Control Association*, **4(1)**, 94-96.

United States Animal Health Association (1992) *Foreign Animal Diseases*. Carter Printing Company, Virginia.

USDA:Animal and Plant Health Inspection Service (1992) African Horse Sickness Emergency Disease Guidelines, 12-18.

USDA:Animal and Plant Health Inspection Service (1994) Foot-and-Mouth Disease: Sources of Outbreaks and Hazard Categorization of Modes of Virus Transmission.

USDA:Animal and Plant Health Inspection Service (1984) Foreign Animal Disease Reference Manual, 175-179.

USDA:Animal and Plant Health Inspection Service (1972) Methods and menas of spread of foot-and-mouth disease. Foreign animal disease report, 8-12.

Van Velden, DJJ; Meyer, JD; Olivier, J; et al (1977) Rift Valley Fever Affecting Humans in South Africa. *SA Mediese Tydskrif*, **51(24)**, 867-871.

Verwoerd, DW; Erasmus, BJ (1994) Bluetongue. *Infectious Diseases of Livestock, Vol. 1*, Coetzer, JAW; Thompson, GR; Tustin, RC eds. Oxford University Press, New York.

Watson, WA (1981) Recent Advances and Concepts of Control. *Can. vet. J.*, **22**, 311-320.

Webster, RG; Sharp, GB; Claas, ECJ (1995) Interspecies Transmission of Influenza Viruses. *American Journal of Respiratory and Critical Care Medicine*, **152**, 525-530.

Weiss, WE (1968) Lumpy skin disease. *Emerging diseases of animals.* FAO Agricultural Studies Bulletin No. 61, 179-201.

Wilson, ML; Chapman, LE; Hall, DB; et al (1994) Rift Valley Fever in Rural Northern Senegal: Human Risk Factors and Potential Vectors. *American Journal of Tropical Medicine and Hygeine*, **50(6)**, 663-675.

World Tourism Organization (1997) *Compendium of Tourism Statistics*. World Tourism Organization, Spain.

Zhou, N; He, S; Zhang, T; et al (1996) Influenza Infection in Humans and Pigs in Southeastern China. *Archives of Virology,* **141**, 649-661.